CURRICULUM VITAE
HOWARD M. SANDLER, M.D.

EDUCATION AND TRAINING:

M.D., University of Maryland, School of Medicine, Baltimore, Maryland, 1977

B.S. (cum laude), University of Maryland, College Park, Maryland, 1973

EXPERIENCE:

1983 - Present	President Sandler Occupational Medicine Associates, Inc. Melville, NY
1982 - 1983	Vice President Corporate Medical Health Director Occupational Health Services, Inc. New York, NY
1981 - 1982	Corporate Medical Director Executive Vice President Industrial Health and Hygiene Group McLean, VA
1981	Medical Officer Office of Extramural Coordination and Special Projects National Institute for Occupational Safety and Health Rockville, MD
1980 - 1981	Medical Officer Office of Program Planning and Evaluation National Institute for Occupational Safety and Health Rockville, MD
1979 - 1980	Consulting Occupational Physician JRB Associates, Inc. McLean, VA
1978 - 1979	Consulting Occupational Physician Equitable Environmental Health, Inc. Rockville, MD

TEACHING/APPOINTMENTS:

American Industrial Hygiene Association/American Society of Safety Engineers
Professional Development Course
Musculoskeletal Disorders, March, 2000

American Industrial Hygiene Association/American Society of Safety Engineers
Professional Development Course
Causation Determination in Occupational Health, November, 1999

Program of Ergonomics and Biomechanics, New York University
Occupational and Industrial Orthopaedic Center
Hospital for Joint Diseases Orthopaedic Institute
Seminar - Occupational Health and Managed Care, May, 1999

National Occupational Research Agenda Meeting
National Institute for Occupational Safety and Health
Work-Related Musculoskeletal Disorders, Invited Participant, Spring, 1999

American Industrial Hygiene Association/American Society of Safety Engineers
Professional Development Course
Musculoskeletal Disorders, February, 1999

Epidemiology of Physical Factors
Invited Participant and Presenter for the Workshop on Work-Related Musculoskeletal Injuries: Examining the Research Base
National Research Council/National Academy of Sciences, August, 1998

Occupational Medicine Residency Program
Occupational Health Division
Environmental and Occupational Health Sciences Institute
University of Medicine and Dentistry of New Jersey
Seminar Series - Causation Determination, March, 1998

Occupational Medicine Section, Fundamental Aspects of Industrial Hygiene - Introductory Course of the American Industrial Hygiene Association, 1980's

126-12-64 WW
SMALL, GARY W

Outpatient Consultation
MEDICINE/Allergy-Immunology

Date of Service: Friday, June 30, 2000

Reason For Consultation: Patient seen with the problem of episodic shortness of breath.

History Of Present Illness: This 48-year-old man, who is the head of the Aging Center here at UCLA is seen because of ongoing problems of shortness of breath. The patient in fact was seen initially in the allergy section in August of 1997. He did have a history of longstanding allergic rhinitis since his teenage years, and had been treated with immunotherapy for cat, dog, dust mites and various pollens. He did not have much problems in Boston but, about ten years ago, he thought he had some episodes of wheezing and was given inhalers which helped, but then the wheezing episodes disappeared and did not recur. When he was seen in 1997, he had the increasing upper respiratory infections with two young children, and a problem with increasing protracted cough. He got less than complete relief by inhalers. Skin testing at that time showed reactivity to cat, some activity to dust mite, and in fact he was on Claritin when this testing was done. At that time, he was suggested to be treated for upper respiratory infections and evaluated for possibility of lower airway disease - asthma. He subsequently saw Dr. Tashkin and underwent a methacholine challenge. He had a negative methacholine challenge in September of 1997, having had normal spirometry in July of 1997. Over the subsequent three years, however, he has had a real good response in that his cough is gone, but he now has episodes of dyspnea. This dyspnea is not sudden in onset. It seems to be worse in the spring and during hot weather, and also may be exacerbated by exercise or sleeping with feather pillows. He does get some seasonal sinus headaches. He has increased the medications now to the point where he is on both Flovent 110 2 puffs twice a day, Claritin 24 once a day, Singulair 10 mg once a day which was started three to four years ago, and he is using Flovent 44 mcg dose in his nose with an adaptor twice a day. He does feel that the inhaled steroids help his episodes of dyspnea, while inhaled bronchodilators have not. He does not note rapid onset on minutes or rapid offset of the dyspnea. It is not associated with frank stridor or difficulty speaking or swallowing. He has had a careful workup with reflux which was negative.

Past Medical History: Is only relevant for intermittent low back pain.

Past Surgical History: None.

Present Medications: Singulair 10 mg a day, Flovent 110 2 puffs twice a day, Claritin 24, Flovent 44 b.i.d. in the nose, and Hytrin 1 mg a day.

Drug Allergies: Keflex which gave him a rash in 1996.

Environmental Survey: He does have dust mite proof covers and dust mite measures in place in the bedroom. He has undergone previous allergy testing, both as noted and three to four months he had tests at the Pulmonary Division that looked moderately positive for molds and pollen.

Social And Occupational History: He is a professor of psychiatry at UCLA and head of the Aging Center. He is married. He is a previous smoker. He used to smoke 2-10 cigarettes a day. He drinks approximately one alcoholic beverage a day, and does not use recreational drugs.

Family History: Is relevant for a positive history for hay fever, diabetes, strokes, thyroid disease and hypertension.

Review of Systems: Complete review for 11 systems can be found in the chart. The only relevent symptoms of note not covered already is mild dandruff of eyelashes.

Physical Exam: Vital signs normal as noted in the chart.

Dictated: 6/23/2000 13:37
By: Andrew Saxon, M.D. (P05738)
Reference number: M1-510233
Transcribed: 6/24/2000 14:32
By: RD:RD/121
Reference number: UCA624M5.PC1
Received: 6/24/2000 15:23
Document ID Number: 451042

Well-developed, well-nourished man who appears his stated age.
SKIN: shows there no generalized rash, bruising or erythema. Mild seborrhea. There are no ulcers, bleeding, or blistering.
HEENT: Normocephalic, Ears canals clear, TM normal, PEERL, EOM full, conjunctivae not injected, sclerae non-icteric. Nose normal with no discharge, polyps, or bleeding. Mouth is clear. Neck is supple. Thyroid is not enlarged. The trachea is midline.
LYMPH: There are enlarged no cervical, axillary, or inguinally lymph nodes.
CHEST: clear to percussion and auscultation
CARDIAC: S1 and S2 are normal. No murmurs or gallops. no peripheral edema
ABDOMINAL: Normal bowel sounds, liver is not enlarged, spleen is not palpable.
EXTREMITIES No cyanosis, clubbing, or edema. No onychomycosis. Motor tone and mass normal
NEUROLOGIC: oriented in all spheres. Mood and affect are appropriate. Reflexes are 2-3+ at the knees and ankles.

Allergy Skin Testing: was undertaken with a panel of trees, grasses, weeds, molds, etc. This showed clear reactivity to walnut, sycamore, olive tree, as well as Alternaria and several other molds. Dust mites were also positive as was cat. Grass and weed pollen was negative.

Assessment: 1) Upper airway allergic disease with allergic rhinitis. 2) Sinus discomfort related to #1. 3) Dyspnea - etiology unknown. 4) Seborrheic dermatitis, mild

Discussion: The main symptom this patient is having of concern to him now is this dyspnea. In view of a negative methacholine challenge, normal pulmonary function tests and no clinical response to bronchodilators, I find it hard to accept this as small airway disease or asthma. His equivocal response to corticosteroids and the fact that he has not been able to wean them is hard to interpret as to whether this is truly an inflammatory process or not. He has no features to suggest vocal cord dysfunction. At this juncture, we will aggressively treat his upper airway allergic disease and then, in about two weeks, we will stop the treatment for potential lower airway disease and carefully evaluate him when he is frankly dyspneic to see if there is any evidence for true asthma versus other etiologies.

Andrew Saxon, M.D. (P05738)
Electronically signed (Mon Jul 10 16:34:35 PDT 2000)
MD5 checksum: f5aed11de9284f0a2fbbef2988321f3f

cc:
Samuel Skootsky, M.D.
200 UCLA Medical Plaza

Gary Small, M.D.
10812 Porte Fino Place
Los Angeles, California 90077

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November 26, 2003

Mr. Scott A. Flinders, Esq.
Emerson & Manke, PLLC
302 E. Carson Avenue, Suite 1002
Las Vegas, Nevada 89101

Report regarding complaints of Mr. Anthony (Tony) Pitaro.
PITARO, ANTHONY
DOB 2.27.59

Dear Mr. Flinders:

Mr. Anthony Pitaro is a 44-year-old man who was born on February 27, 1959. He currently has a number of medical conditions and issues. I have outlined these medical issues and provided and analysis of them below. In the end, you will see that if mold has any adverse effects on his health, it would have been a minimal one and in the setting of his other medical problems, one that would essentially be trivial.

Data Base:
I have reviewed the extensive medical records (over 5000 pages) on Mr. Tony Pitaro that span the time frame of September 1995, through February 2003. These medical records are listed in more detail in your letter to me of June 17,2003. In addition, I have also read the depositions of Drs. Singh and Keene. I have also reviewed environmental testing that was done in May 1998 in the office where Mr. Tony Pitaro worked.

Opinions regarding Mr. Pitaro's medical condition.
Overall, the Mr. Pitaro's complaints can be broken down into several major categories.

Myasthenia Gravis with associated benign thymoma, resected. Mr. Pitaro's overarching medical problem is myasthenia gravis (MG). I will let Dr. Alexander, the neurologist, give you the details of this disease. MG is an autoimmune disease in which autoantibodies are made against the motor end plates. This leads to disruption of nerve conduction and muscle weakness. His MG was associated with the presence of a benign-nonmalignant tumor of the thymus--a thymoma. There is a well-recognized association between the occurrence of this benign thyroid tumor and of MG. Mr. Pitaro had a particular form of this involving the throat area, so called bulbar form of this disease. The diagnosis of myasthenia

gravis was not appreciated until a neurologist saw him in August 1998. However, I feel it is important to recognize for his health issues that this has likely been going on for quite some time prior to its recognition by his doctors. . As I will discuss later, his MG and the particular form of it he has is a very important factor in his respiratory symptoms. Subsequent to his recognition of myasthenia gravis, he has been treated with a variety of immunosuppressive drugs including corticosteroids and Imuran. There is no reliable medical literature to support the notion that mold exposure such as may have occurred in Mr. Pitaro is associated with the induction (cause) or exacerbation (worsening) of autoimmune disease in general, and specifically, there is no reasonable suggestion anywhere in the medical literature that I am aware of, that mold exposure will induce or worsen myasthenia gravis. Furthermore, there is no medical literature that would support the hypothesis that mold exposure associated with the development of benign thymomas.

Sleep apnea. He has a history of sleep apnea, which was documented in 1995. His obesity at that time would have been a major contributing factor; being 6 feet 1 inch tall and having weights go as high as 280 pounds. Obstructive sleep apnea is an anatomical issue, which is unrelated to mold exposure in any aspect. Sleep apnea can have a variety of adverse health outcomes, the most common of which would be generalized symptomatology of fatigue, malaise, and daytime sleepiness. It also can lead to headache as well as more serious medical complications such as heart disease, hypertension, and lung disease. The current status of his sleep apnea is unclear, and thus it may be playing an important role in some of his present symptomatology. I don't see that he is currently being treated with CPAP and whether he has ongoing problems with obstructive sleep apnea, e.g. he as not had a study recently to determine the present level of this problem. He has had surgery for this and has had weight loss, which would be an important factor in dealing with obstructive sleep apnea. Sleep apnea is an anatomical issue that is generally completely unrelated to mold exposure.

Respiratory problems. The records from 1995 reflect a history of having had seasonal allergic rhinitis (hay fever) as a child. He also gives a history of having several episodes of pneumonia, once as a child and once in the early 1990s. He had pulmonary function tests in September 1995, which showed evidence of obstructive lung disease with an elevated diffusion capacity when corrected for alveolar ventilation. These findings are suggestive of reactive airway disease, although no bronchodilator response was done. He did have spirometry then in December 1996, which showed both moderate restrictive disease a significant bronchodilator response, but here the baseline spirometry is invalid due to artifacts so these tests can not be used to definitive establish the diagnosis of reactive airways disease. Notably, at this time, these were concerns of postnasal drainage and possible acid reflux and silent aspiration. Indeed, in March 1997, it was discovered that he had pansinusitis, a condition that would lead to chronic postnasal drainage and drainage of mucopurulent (infected) materials. He underwent functional endoscopic sinus surgery for this in April 1997. However, he had persistent problems with a cough through 1997 when ultimately be a diagnosis of myasthenia was established.

It is my opinion that his bulbar myasthenia has played the critical important role in his respiratory symptoms. This key role for myasthenia gravis is related to both a) direct neuromuscular weakness from myasthenia gravis and b) chronic micro aspiration secondary to his unrecognized bulbar myasthenia. Thus myasthenia gravis would be the major driver in his respirstory symptoms in the 1995 onward time frame. Neuromuscular weakness itself will lead to a decreased lung volume and decreased flow as seen in Mr. Pitaro on his pulmonary function tests. Microaspiration will lead to marked irritation of the upper and lower airway with changes of lung scarring as seen in Mr. Pitaro. This problem was worsened by the presence of his chronic sinus disease as in individuals with chronic sinusitis such as Mr. Pitaro; there

is ongoing postnasal drainage of mucopurulent material In a patient such as Mr. Pitaro with bulbar myasthenia, this postnasal drip of mucopurulent materials is are more likely to lead to significant lower respiratory problems due to the nature of the material being aspirated. The nature of the changes in his lungs which involve scarring the dependent areas (lower parts), also is strongly supportive of the idea that a chronic, unrecognized microaspiration is the primary cause for his respiratory problems.

The records also reflect a history of "dust mite" allergy and "seasonal allergies." He has had pet animals such as dogs in the past. I am not aware of his current status regarding having pets in his home. The records that he had allergy prick and intradermal skin testing on 2.16.98. These showed Prick and Intradermal testing that was negative for grasses, trees, weeds, Alternaria, Cladosporium, and "mixed fungus". Intradermal shows a 2+ reaction to dust mite. Thus, the only allergen one can define Mr. Pitaro as being possibly allergic to is dust mites, particularly given that intradermal testing was negative - testing that is very sensitive. While, some have opined that Mr. Pitaro's asthma would get worse due to exposure to Stachybotrys, I so not agree with that opinion. Stachybotrys is NOT a relevant airborne allergen as the levels of this mold in air are generally so low as to be inconsequential as an allergen. This is in contrast to molds such as Cladosporium and Alternaria outdoors or Penicillium and Aspergillus indoors. In this regard, we have no airborne sampling from the office environment in question.

He has evidence of having had chronic sinusitis, a very common chronic conditions present in adults in our country. Chronic sinusitis is an inflammatory disease of the sinuses where there is bacterial and/or allergic inflammation. Mr. Pitaro's risk factors for this would be allergy and his deviated septum as these lead to obstruction of the sinus drainage, the key element in the development of chronic sinusitis. Chronic sinusitis is not due to mold infection in spite of a much cited single article that appeared in the Mayo Clinic Proceedings in 1999 and certainly, there is no such evidence that Stachybotrys causes sinusitis.

Overall, the nature of his lung disease is multifactoral with his myasthenia gravis being the key factor leading to neuromuscular weakness of both the lungs (chest wall) and the swallowing mechanism. The latter gave rise to recurrent chronic microaspiration that has led to the more damaging aspects of his respiratory illness. He may be allergic person but his allergies, if present, are a minor issue and they do not include allergies to mold

I do note that he has grown Candida (a yeast) from his respiratory tract one at least one occasion (3.28.08) and an unidentified yeast (likely Candida) on 1.25.02. This is completely unrelated to any environmental exposure to mold, fungi or yeast. Such yeast is present in the mouth of the majority of human beings as commensal organisms. Mr. Pitaro was treated with a large number of antibiotics as well as inhaled and later corticosteroids, each of which will lead to fungal overgrowth. Thus, the explanation for the culturing of yeast from his sputum is simply that it is a reflection of the medications he was given and is completely unrelated to any environmental exposures he may have had to mold or fungi. Review of Mr. Pitaro's records show that he has never had any evidence of any invasive fungal disease. He was evaluated by Dr. Christenson who tried to determine if Mr. Pitaro has any underlying immune deficiency, which would make him more at risk for a fungal infection. This evaluation, done in March 1998 (prior to his immunosuppressive drug treatments), was completely normal.

Dr. Singh has given the opinion that Mr. Pitaro may have hypersensitivity pneumonitis (extrinsic allergic alveolitis due to Stachybotrys. I find this to be an inappropriate suggestion. Hypersensitivity pneumonitis requires high-dose inhalation exposure to foreign proteins. Not only am I unaware of any

case of proven hypersensitivity pneumonitis from Stachybotrys in any setting, but also it would be extraordinarily unlikely to occur in any home or office setting. Stachybotrys is known to be very poorly aerosolized and levels such as would be required for hypersensitivity pneumonitis to occur have *never* been reported in a setting such as Mr. Pitaro worked in. Stachybotrys was found only *in air sampling* in 6% of homes surveyed and the 95% confidence interval for those positive homes as 12-95 spores/m^3.

Dr. Singh also opines about Mr. Pitaro developing an entity that Dr. Singh calls "multiple chemical sensitivity". Multiple chemical sensitivity, now known as idiopathic environmental intolerance, is a psychological belief system that has no basis in physiology. This has been extensively reviewed and critiqued in the medical literature. Exposure to molds or mold products does not lead to this condition of idiopathic environmental intolerances. I have attached the position statement of the American Academy of Allergy, Asthma, and Immunology regarding this subject.

Reactive depression. The records reflect that he appears to have developed reactive depression in 1998 related to the serious health problems he has had. There is no mention of this more recently.

OUTLINE OF MEDICAL RECORDS REVIEWED

Date	Page	Content
9.20.95	623	Spirometry shows FVC of 4.60, FEV1 of 3.75 and FEF25/75 of 2.1 that are 86, 74 and 46% respectively. Normal diffusing capacity.
11.6.95	392	Polysomnography shows sleep apnea - mixed positional, with arousals. Prolonged periods of apnea-free sleep, but not on his back. Periodic leg movements with sleep with episodic arousal, fragmentation and disruption of nocturnal sleep.
12.11.95	1	ENT exam for sleep apnea since 45-pound weight loss. Some intermittent headache. Exam is normal. Mild hypertrophy of turbinates. Slightly redundant and elongated uvula. No suggested anatomic reason for his apnea, other than he is extremely overweight. Weight loss recommended.
12.11.95	113	Patient history form says yes to kidney problems and he circles high blood pressure. No to other problems, including asthma. Does say he has had breathing problems. He is here for sleep apnea. Does not smoke. Wife says he stops breathing at night. Says he has been diagnosed previously with hay fever and pneumonia. Hay fever is seasonal and pneumonia as a child and three years ago as well. Does have a pet dog. Symptoms made worse by wind, dust, air pollution, steam, and tobacco smoke. Works as a private investigator.
1.13.96	2	Discussed with wife sleep apnea positive and patient would like to try CPAP
12.10.96	384	Normal chest x-ray.
12.12.96	313	Normal respiratory flora on sputum culture.
12.12.96	620	Dr. Singh, Pulmonary Associates consult said had pneumonia as a child and hay fever, three years ago having seasonal hay fever, takes Vancenase, and he generally feels miserable. Told he had mild sleep apnea, says he has some acid reflux. Exam is unremarkable. Chest x-rays from earlier in the month are unremarkable. Says spirometry shows severe obstruction with significant bronchodilator improvement. Suggests that his bronchospasm is a result of postnasal drainage and possible acid reflux and silent aspiration.

12.12.96	394	Spirometry shows FVC of 2.31 at 41% and FEV-1 of 1.56 at 34%. There was a 25% and 22% increase post bronchodilator. Peak expiratory flow rate is 6.5, both pre- and post bronchodilator about and these are about 60% of predicted which is 10.14. FEF 25-75 is only 0.9, which was 17% of predicted. Severe obstructive defect.
2.11.97	308	Negative Cocci IgG and IgM.
2.11.97	401	Completely normal CBC, diff, CHEM panel, including lymphocyte percent. Alk phos slightly low at 37.
2.11.97	4223	Chest x-ray shows some improvement in changes in the right base, stranding in the lower lobe. Emphysematous changes in the apices of both lung fields are noted.
2.13.97	380	CT of chest shows shotty adenopathy in the mediastinum. Largest lymph node 1 x 1.5 cm. Some small densities in the right lung are 2 mm with some scarring at the left lung base. No evidence of thymoma or mass.
3.18.97	378	Sinus CT shows total opacification of maxillary sinuses and pretty much of ethmoids, sphenoids and frontals in trouble, too. Confluent masses suggesting polyps in the middle meatus. Diagnosed with pansinusitis and obliteration of the middle meatus with probable polyps.
3.20.97	3	ENT doctor reported he had frequent bouts of pneumonia over four to five months. Feels related to his postnasal drip and cough at the same time frame. On Atrovent, Proventil, Vancenase, Flovent, Claritin, Pepcid and Flonase. Upper respiratory exam just shows some congestion. CT will be reviewed.
3.26.97	3	Sinus CT showed severe intranasal and intrasinus disease. Hardly any air in the sinuses. Suggest he needs septoplasty and functional endoscopic surgery.
3.31.97	377	Chest x-ray shows prominent bronchovesicular markings.
4.4.97	26	Functional endoscopic sinus surgery with bilateral maxillary antrostomies and bilateral ethmoidectomies and septoplasty.
4.4.97	4502	Operative report for functional endoscopic sinus surgery.
4.14.97	3	Postop period endoscopic clean-out in the office was performed today. Refill his Vancenase and pain medicines.
4.21.97	4	We cleaned out nose after surgery from April 4, '97. Large amounts of debris were cleaned out.
5.7.97	7	ENT doctor notes greenish drainage from sinuses. Coughing now for seven months. Put him on Levaquin for Pseudomonas treatment and follows up.
5.28.97	10	Still coughing, very productive with yellow phlegm. Now postnasal drip. He says his chest is congested when he lies down. Upper airway exam by ENT doctor appears well and wants secondary opinion on his chest when he comes back in three more weeks and he does not need any more cleaning out in the small perforation of the septum and only has a minor cough.
6.12.97	367	Pulmonary consult for persistent cough. Says he got sick in October '96 after getting a respiratory infection. A month later he had pneumonia and was treated with antibiotics and had persistent cough. Since then he had persistent bouts of postnasal drip and sinus problems. Cough persists. Has moderate restrictive defect with severe reduction in his FEF 25-75. Showed mixed positional sleep apnea, 95 on testing, with some peribronchial thickening and is felt to have asthma, chronic sinusitis, sleep apnea, moderately overweight.
7.2.97	11	Doing now well with drainage for the last three to four days. Put him on Augmentin.

7.8.97	387	Spirometry shows FVC 3.41 at 60%, FEV-1 at 2.21 at 49%. FEF 25-75 is 1.13 at 25%. PEF 6.9 and FIF 5.71 and flow loop shows clear obstructive pattern. Weight is 231.
9.24.97	11	Biopsy for Wegener's of nasal cavity.
9.24.97	13	Nasal biopsy shows acute, subacute and chronic inflammation. No fungal forms identified. Said will do deep resections to look for vasculitis. These changes seen are equivocal for focal vasculitis. Suggested getting an ANCA and the deeper sections on page 14 simply show similar features. No discrete vasculitis.
9.24.97	34	Sed rate 8.
9.24.97	35	ANCA less than 1:20.
12.16.97	302	Normal CHEM panel other than slightly elevated LDH and low alkaline phosphatase, which he always has. Differential though is 16.5% lymphocytes. Normal thyroid function tests including TSH.
12.16.97	4229	Says he is seen for persistent cough. His weight has dropped from 275 to 229 since December '96. Given Zithromax. This is for persistent cough.
1.4.98	4070	Admission history for hypoxia with pO_2 of 53, white count of 16,600 and rule out pneumonia, otitis media on the right, probable asthma, chronic sinusitis. Says he has a history of recurrent pneumonia which initially began about 18 months ago in November of '96. Now presented with cough, chest pain, fever the night before.
1.6.98		Sputum shows moderate growth H. parainfluenza and normal flora.
1.15.98	299	No AFB isolated after six weeks and none seen on fluorescent smear.
1.15.98	320	Frozen sections from lymph nodes and mediastinal biopsy are almost pure population of T-cells which are both CD4 and CD8 positive, characteristic pattern of a thymoma. Histology substantiates that. On page 324 is the lymph node biopsy immunophenotyping which essentially looks normal.
1.27.98	537	Liver ultrasound shows small cyst.
2.12.98	297	Sputum culture says normal flora.
2.16.98	518	Weight 220. Physical exam says normal. Given Hycodan for cough. Apparently is on Atrovent and Ocean Nasal Spray. These are nasal.
2.16.98	534	Prick and ID test negative for grasses, trees, weeds, Alternaria, Cladosporium, mixed fungus. Intradermal shows 2+ to dust mites. Antihistamine is 2+.
2.17.98	521	FVC 2.67, FEV1 2.21 with no change with bronchodilators.
2.19.98	293	Completely normal CHEM panel other than slightly elevated GOT and GPT. CBC and diff normal, including lymphocyte percent at 31.2. Normal number of CD19, CD3, CD4 and CD8 cells. Also normal NK cells. HIV antibody is negative, including HIV by PCR.
2.19.98	533	White cell phenotyping shows 31% lymphocytes of a total of 5.29 with normal numbers and distribution.
3.3.98	519	Spirometry shows FVC 2.37, FEV1 1.69 at 42 and 37% of predicted. Increase in FEV1 to 1.93, which is a 14% change. Look at curves.
3.4.98	290	IgA 401, IgM 95, both normal, normal alpha-1 antitrypsin, sed rate 2, C4 24, C2 normal, normal CBC and diff and he is not lymphopenic at this point with 34.7 lymphocytes. PHA, pokeweed, and ConA stimulation were all greater than 70% - normal.
3.6.98	15	Limited CT of the sinus shows diffuse sinusitis with just some air in the left frontal and relative clearing of the sphenoids. Diffuse opacification otherwise.

3.15.98	16	Laryngoscopic exam shows paralyzed left true vocal cord. Apparently had surgery three months prior to remove a mediastinal mass.
3.25.98	17	Says mass removed from neck came back as a thymoma. Has been hoarse since then. Has had Candida growing out of bronchoscopy for which he was started on Diflucan. Says allergy testing was done which was negative, except for "some dust mites." Presently on Flovent, albuterol, Flonase and Atrovent. No reflux symptoms reported. He says he does have left true vocal cord paralysis, but the arytenoids appear to be making attempts, there may be only a temporary paralysis.
3.31.98	538	Dr. Christiansen wrote a letter to Dr. Lobello saying the first pulmonary function test did not respond to bronchodilators. On the second one he did have a change of 15% and he says this is just shy of the ATS criteria, but I think he can be given a diagnosis of asthma. Says improved by the corticosteroids, stopped because Candida was grown from his respiratory tract. He poses the questions that are before the court as: "1. Does he have subtle immune deficiency that is sometimes associated with thymoma? 2. Is there a reason for his recurrent sinusitis? 3. Are all these related to his new onset asthma? 4. What is the etiology of this cough which started all this in the first place?" Note that he has lost 70 pounds. It is not intentional. I think he may have clinical depression. "I would if I were in his shoes."
5.5.98	39	Discharge summary says he has a history of bronchitis, chronic sinusitis, hypoxemia - resolved, asthma, history of thymoma resection - probably benign, previous sinus surgery and nasal polyp. Admitted for shortness of breath with pCO_2 of 38, pO_2 of 48. Given ceftazidime, Unasyn and steroids. Given treatment for his asthma. Discharged much better. Clear sinus discharge which got better in time. Admitted with a white count of 16,500 and less than 1% eosinophils and 90% neutrophils.
5.12.98	33	Normal CBC including white count at 9.5 and leukocyte count of 34.9.
6.10.98	30	Aminophylline level 16.9.
6.19.98	286	Sputum shows no pathogens.
6.19.98	653	Dr. Singh says that he has asthma, sinusitis and toxic mold exposure, says that spray painting was being done by his wife and made him worse. His doctor's impression is that he had sinusitis, asthma and toxic mold exposure to Stachybotrys atra with consequent multiple chemical sensitivities (MCS). He says, "As a result he is now sensitive to multiple chemicals that are nonspecific and on windy days with sprays and strong odors he has increased bronchospastic attacks, though his skin testing does not show any significant positive reaction."
7.8.98	602	Craner consultation, says referred by Dr. Eckhart Johanning in New York, was employed in an office space in one of the rooms of his brother's law office at 815 East Third Street in downtown Las Vegas, presently on Theo-Dur, Singulair one a day, Flovent 220 two puffs b.i.d., Flonase, potassium, albuterol tablets two a day and Allegra two a day. Has one dog. And his diagnosis is chronic sinusitis/occupational. Asthma secondary to sinusitis versus allergic; Occupational, and history of benign thymoma with iatrogenic vocal cord paralysis; Non-occupational. Says he had the symptoms related to his exposure and got better when he wasn't there. Craner says he did not start to get better until he left the work environment. He says the etiology is most likely exposure to one or more molds. Says mycotoxins or molds can produce irritant, neurologic,

		constitutional and dermatologic disorders. Says it was likely due to allergic inflammation with inadequate drainage. Says his prognosis is good.
8.24.98	694	Dr. Goodman, neurology and neurodiagnostic testing. She saw him in '92 for a post concussive syndrome. He complains of fatigability, frequent napping, difficulty with his speech for the last three weeks. Tongue does not make words. Visual dysfunction last month for focusing or occasional diplopia. Feels possible myasthenia.
9.9.98	4966	MRI of brain. Brain normal, marked sinus disease noted.
9.23.98	19	Says his neurologist sent him back because his sinuses were full again. Plans to do another fiberoptic endoscopy surgery on the sinuses.
10.7.98	876	Neurology followup is much worse with his dysphagia. His Mestinon not helping as much. Short of breath at night. Hip and leg pain. Severe dysphagia on exam. No notation of changing his medicines.
10.15.98	692	CT of chest. There is a 3.5 x 3.5 cm right superior mediastinal mass. He says this is new since the study of 02/13/97. Some atelectasis in the right middle lobe.
11.16.98	282	Normal thyroid studies including TSH.
11.19.98	278	Sputum: No pathogens isolated. Many white cells.
11.23.98	315	Path Report: A 10 x 4 x 5 cm lobulated mass of fat within which is a 6 x 6 x 4.6 gray area, a noncapsulated lobulated lesion of uniform small mature lymphocytes with scattered Tingible body macrophages felt to be lymphocyte predominant lymphoma. Pending immunohistochemical stains. His special studies - stains show very strong Leu-7 binding as well as positive pankeratin staining of large epithelioid cells.
12.1.98	1325	Discharge summary. Admitted for myasthenia, thymoma and reactive airways. Underwent plasmapheresis, some anxiety-related chest pain not cardiac in origin. Had surgical removal of his thymic mass with a median sternotomy. And the operative report of the sternotomy with resection is on page 1328 from 11/23/98.
12.1.98	3475	Dr. Lamancusa discharge summary. Says Anthony is a high school classmate of his. Says he developed chronic sinusitis that was "felt to be related to mold exposure work". Had sinus surgery with little help. Had a chest mass found in January and exploratory surgery and was found to have a thymoma. However, only about a third of the thymoma was removed; and subsequently, he was well until August of '98 when he developed neurologic problems. He underwent plasmapheresis, thymectomy, and was discharged home.
12.9.98	674	Surgeon who did thymectomy. Says no sign of infection. His Mestinon is cut down to one tablet twice a day. Incision healing well. Lungs are clear. A little bit of hemoptysis cleared after he stopped his anti-inflammatory - nonsteroidal.
12.15.98	1324	Neurology note saying he was in the hospital from November 16 to December 1, '98. He said he was given plasmapheresis from November 16 to the 20th and then on November 23, '98, he had a thymectomy.
12.15.98	274	ACR binding 13.3.
12.23.98	21	Sinuses show some thickening and possibly polyps in the maxillaries. Ethmoids show marked disease, mucus thickening in the frontals as well as sphenoids.
2.4.99	28	Endoscopic surgery. Preoperative diagnosis is chronic mold sinusitis, myasthenia gravis and recurrent sinusitis. It says that he has a protracted history of sinusitis which "was eventually diagnosed with a Stachybotrys ultra (sp) sinusitis which itself only causes problems with his lungs and his sinuses."

3.12.99	1319	Neurology followup. Begins him on Imuran in view of recurrent symptoms and rising ACR antibody level. Says he was diagnosed in fall of '98 and he was known to have a thymoma by history, and that he underwent a thymectomy as well as a plasmapheresis in November '98.
5.7.99	1315	Stable on 180 mg long-acting Mestinon and 90 mg regular twice a day plus Imuran 150 mg.
6.9.99	266	Normal CBC, CHEM panel except lymphopenia is 15%. ACR binding 5.1.
6.13.99	360	IME by Dr. Willoughby at the Lung Center of Nevada. Underwent total thyroidectomy, it says, in 11/98. Underwent plasmapheresis afterward and was treated with Mestinon as well as Imuran for the past three months, it says. Also on albuterol, Claritin, Flovent. His impression is asthma, pre existing prior to Stachybotrys exposure, but exacerbated by Stachybotrys. Myasthenia gravis with muscle weakness in the respiratory muscles and bibasilar pulmonary atelectasis. Chronic sinusitis and nasal polyps controlled with surgery and chronic cough secondary to asthma, myasthenia and sinusitis. Says that, "he has had asthma before the exposure according to his pulmonary function tests from 1995," but that "his asthma was clearly worsened by his Stachybotrys exposure," but improved when he removed himself from the environment and "there is no conclusive evidence that it was permanently by the exposure." "Many of his symptoms at this time are secondary to myasthenia gravis with muscle weakness and not to asthma, Stachybotrys or sinusitis."
6.24.99	385	Spirometry shows moderate restrictive disease by helium total lung capacity with moderate reduction in DLCO at 56% of predicted when adjusted.
7.26.99	24	Has resolving oral thrush, resolving otitis externa. Stop the Levaquin and restart Ceftin, Diflucan as well. Apparently he called somebody about using gentamicin with the Astelin spray as a topical treatment and apparently they felt that would be fine.
9.9.99	259	Normal CBC except for lymphopenia at 18.9% of 8.1. ACR binding 4.7.
9.16.99	25	Says he had a reaction to gentamicin and Astelin caused him a bloody nose so he stopped those and is just using saline.
10.1.99	253	Normal CBC and diff except lymphocytes 22.5%. ACR binding 13.1.
11.16.99	1311	Neurology followup. Had stopped Imuran but restarted at 100 mg a day. Had stopped for infection. Feels like he had had Mestinon long-acting.
12.9.99	1304	Barium swallow shows poor epiglottic function with large amounts of pooling in the piriform sinus and vallecula with large amounts of residua, pharyngeal penetration with solids.
12.10.99	1306	CT of chest: No mediastinal mass or adenopathy, small amount of atelectasis, right upper, right middle and lower lobe on left.
12.19.99	2135	Discharge note for exacerbation of myasthenia gravis and respiratory failure. He received three doses IV IG on December 7 through 10, plasmapheresis from the 10th to the 14th, and intubated from the 12th to the 15th. Apparently he admitted that his speech is getting worse. He was put on prednisone and stopped the Imuran because of some liver changes. Ptosis, dysarthria and dysphonia were noted and he was admitted. He was discharged on Mestinon 180 a day, Imuran 100 a day and Lortab. The neurologist felt he was having an exacerbation of myasthenia - 2138.
2.5.00	242	Normal CBC and diff except 18.6% lymphocytes. ACR 14.9.

2.22.00	2850	Admission history. Having increasing weakness and shortness of breath. Plasmapheresis was begun. Father is reported to have died from Guillain-Barre syndrome. Treated for upper respiratory infection with Zithromax and pulmonary therapy.
2.22.00	2852	Neurology consult. Here it says he had mold exposure in the early '90s that had resulted in chronic sinusitis as well as lung infection. Says he had a thymectomy in November of '98. He says exacerbation of myasthenia despite thymectomy, IV IG, Solu-Medrol and plasmapheresis and also history of asthma.
3.2.00	236	CBC normal except lymphopenia at 14.8 [later one may be in error]. ACR binding 14.7.
3.5.00	235	Sputum shows heavy growth Strep. pneumoniae.
3.10.00	2971	Discharge summary. Complaining of shortness of breath, weakness of his extremities and head lag, was found to be hypoxic and have generalized weakness and admitted. He had plasmapheresis and got markedly improved blood gases. It was felt that the Zithromax may have been exacerbating his myasthenia. At time of discharge he was back to normal. Maintained on Imuran and prednisone.
3.13.00	4262	Pulmonary followup for this week. Vital capacity is 2.65 and FEV1 is 1.69, and he more restricted than obstructed. Infiltrate had cleared in the left.
3.27.00	230	Normal CHEM panel except globulin is elevated at 5.4. CBC and diff shows mild anemia at 33.5 and he is not macrocytic and his lymphocyte percent is 27. ACR antibody 16.7.
3.29.00	228	PO_2 66, pCO_2 36, pH normal.
3.30.00	950	Keesey consult at UCLA, says that myasthenia symptoms first occurred in 1998 in July, says the thymoma was first noted in January '98 during the pneumonia. Says the patient tells him it tripled in size in '98 and was removed. Says he had an MRI a week before, which was negative. Had some facial weakness and slurred speech, but negative, and his inspiratory force was normal at greater than -60. Limbs are strong. Impression is: Has a history of severe oropharyngeal myasthenia which was doing better, presently on immunosuppression and IVIG. Says he has been on adequate dose of Imuran since December. Liver enzymes are now normal. I would continue the Imuran, he says. Tells him he can increase his Mestinon. Suggests giving IVIG treatments every three weeks instead of every week, but give him three in a row. Says monitor potential for thymoma recurrence.
5.5.00	2052	Jarvis, Dr Joseph Q. MD MSPH report. His assessment is 1. "building related asthma, rhinitis and sinusitis. 2 Consider building related hypersensitivity pneumonitis 3. Thymoma-status post resection of thymus. 4. Myesthenia Gravis secondary to thymoma." He then says the amount of mold in the office is unclear though several sources lead him to " suspect a greater area of overt fungal growth and should be tolerated according to the New York guidelines". He says that new onset rhinitis, asthma and hypersensitivity pneumonitis has been repeatedly observed he alleges. He says that outbreaks of asthma and hypersensitivity pneumonitis in water damage buildings have been described in medical literature and he says allergic chest disease is accepted as one of the only few potentially serious medical complications related to poor indoor air quality. Says no one has even considered hypersensitivity pneumonitis and he thinks it might be

		hypersensitivity pneumonitis. He says that the building related illness is unrelated to his Myasthenia Gravis.
5.8.00	266	Lymphopenia at 15.7% of 8600 and Acetylcholine receptor binding 11.4.
5.16.00	928	Eye exam says chronic open angle glaucoma suspect based on optic nerve findings.
6.14.00	4263	To Whom It May Concern letter from Dr. Singh. Says, "This patient has had Stachysboira (sp) exposure. He is permanently disabled from a pulmonary standpoint because of his previous chronic exposure. I concur with Dr. Jarvis's opinion that Mr. Pitaro has hypersensitivity pneumonitis". As a result of this he is permanently disabled from a pulmonary standpoint."
9.20.00	216	Normal CHEM panel. Acetylcholine receptor 12.9. CBC and diff normal except decreased lymphocytes percent at 10. Slight macrocytic changes. This findings, then, are repeated over and over again for the next couple of years. – turns out likely to be drug related – Imuran and prednisone
8.6.01	196	Completely normal CHEM panel. CBC and diff is normal other than slight macrocytic changes and lymphopenia. They now repeat this test just about monthly for well over a year and just about always the same. Acetylcholine receptor binding 14.7.
10.4.01	4275	Chest x-ray shows bilateral lower lung field fibrotic changes, appear to be exactly the same as before, thus stable and chronic. No new lesions.
12.3.01	4286	PFT's show FVC of 3.19 and FEV1 of 1.73 at 59 and 34% of predicted and no change with bronchodilator with obstructive restrictive pattern.
12.07.01	188	CHEM panel all normal except minimally elevated liver enzymes. Blood count shows macrocytic indices and lymphopenia at 9%. Acetylcholine receptor binding 15.8 and this is again repeated on 9/21/01 as well.
1.25.02	182	Sputum culture shows final growth is light yeast, moderate growth normal upper respiratory flora.
2.28.02	179	Normal CHEM panel. Liver function, hemogram normal, except for lymphopenia at 7.7%. Acetylcholine receptor antibody 23.4.
6.20.02	175	Acetylcholine receptor antibody 20.1 with macrocytic indices on CBC and 60% lymphocytes of normal number.
8.6.02	170	CHEM panel shows slightly elevated fasting glucose and liver functions mildly elevated. This is for the first time, but just above normal. GPT 49, GOT 55 and LDH 295. Again, has macrocytic red cells and lymphopenia at 5%. Acetylcholine receptor binding is elevated at 6.7.
8.21.02	966	Notes that he is slightly macrocytic. Lymphocytes are low at 4.8%. Says elevated LDH due to prednisone, neutropenia secondary to prednisone, Imuran, myasthenia gravis status post total thymectomy. Gives him a B12 injection, cuts down his prednisone to 7.5.
9.19.02	161	Acetylcholine receptor binding 6.2.
10.21.02	154	Normal hematocrit, but again macrocytic indices and normal white count at 9.4, but only 6% lymphocytes. Acetylcholine receptor antibody 6.3 with normal to 0.7.
10.21.02	158	Normal TSH and thyroid.
12.18.02	150	Normal CHEM panel. Elevated vitamin B12. Acetylcholine receptor antibody positive at 10.2
2.21.03	146	CHEM panel essentially all normal other than low alkaline phos. Liver panel all normal, cholesterol 217. T4 normal. ACTH-binding antibody 10.1 with normal less than 0.7. White count normal with only 12% lymphs and 81% neutrophils.

Macrocytic change with increased MCV and MCH at 106 and 35.5. Not anemic with hematocrit of 43.9.

ENVIRONMENTAL

Exposure in office primarily 9.96 to 5.98. There were some surface sampling done in May 1998 which showed minimal growth except for one Stachy on the upstairs storage ceiling tile. No results of airborne sampling

b

5.18.98	617	ENVIRONMENTAL. Direct surface assessments at Tony's office are essentially minimal growth except for one Stachy on the upstairs storage ceiling tile. Tyro HVAC return filter boxes negative, as are the others essentially.
5.28.98	649	ENVIRONMENTAL. Letter re Site visit about the building on 5.18. Says took some tape, bulk and swab samples.

Sincerely yours

Andrew Saxon, MD

Andrew Saxon, MD

Professional Activities

- Are you a full-time professor at UCLA?
- Do you have any professional activities which result in income generation for you outside of your official position at UCLA?
- Did you file federal tax returns which show income from a source other than UCLA in the past 10 years? (Request copies of tax returns from last 3 years to dispute this)
- Would financial documents from your department at UCLA reflect all of the billable work and income you would have generated from consulting activities in litigated matters?
- What is your hourly rate for expert consulting? Testimony?
- What percentage of the income generated from such consulting comes back to you personally as income?
- Would your contract with UCLA reflect how much income you are entitled to as a result of non-salary income generated from medicolegal consulting?
- In how many cases in the past 2 years have you, in your capacity as professor at UCLA or otherwise, been retained or paid by a law firm, insurance company, or other entity representing a defendant in a lawsuit involving alleged exposure to or adverse health effects from mold? (He will probably claim he doesn't know...will probably lie or withhold information). Then:
    - Were you retained as an expert in the matter of Anthony Pitaro in Las Vegas, Nevada? Were you deposed in that matter? (6/9/04)
    - Were you retained as an expert in the Spectrum Condominiums litigation (Santa Ana, CA/Roberston)?
    - Were you retained as an expert in the matter of George Allison and Lurley Winton v Wel Del Amo Pacific Condominium Association (? Were you deposed in that matter? (2/1/02)
    - Were you retained by the firm of Lorber Penguilly in Las Vegas to work on the Pahrump Justice Complex case? (2002-2004)
    - Were you retained in the matter of Shamiram Chandon v Peninsula Plaza HOA (San Mateo Co., CA)
    - Have you examined Dr. Gary Small and/or his wife? Do you know Dr. Small? (Professor of Psychiatry at UCLA; big mold problem.)
- (Demand copies of all deposition transcripts for the past 5 years, or list of cases.)
- Altogether for how many years have you been doing expert work or "independent" medical evaluations on mold cases?
- In how many cases have you been retained by the firm of Lewis, D'Amato, Brisbois & Bisgaard (LA)?

- Of the (x) legal cases that you have consulted on regarding mold exposure in the past 5 years, what percentage have been on behalf of a defendant? (100%) Plaintiff? (0%; or will claim he doesn't recall)
- How many individuals as patients have come to you or your clinic, that you have examined, who have non-litigated health complaints or concerns about mold exposure? Would this information be available through scheduling and patient billing information at UCLA?
- Have you been consulted or retained as an expert to investigate a building(s) with suspected or known indoor mold contamination?
- Please list all of the plaintiffs you have examined in connection with a mold exposure claim in the past: 90 days; 180 days; year; 2 years.
- Is it fair to say that your litigation consulting work in the area of indoor mold is highly skewed toward work for defendants?
- Is it the policy of UCLA to take work only on behalf of certain parties in litigated matters?
- Is the Dean of the UCLA School of Medicine aware of your professional consulting/expert activities? How about the Board of Regents of the University of California?

ACOEM Position Paper

- When were you contacted to participate in the preparation of the ACOEM "Evidence-Based Statement" on mold (referring in this line of questions to "Adverse Human Health Effects Associated with Molds in the Indoor Environment").
- Who specifically at ACOEM contacted you? Or were you approached by Dr. Hardin or Dr. Kelman?
- Who funded your participation in the preparation of this document? Did ACOEM pay you or UCLA for your services? If yes, how much?
- Are you a member of ACOEM? (No)
- Do you have any training in the specialty of Occupational Medicine? (He may attempt to say he does; if so, ask for specific training, documentation, certifications)
- At the time the document was written, was Dr. Kelman or Dr. Hardin a member of ACOEM? (No) Is either of them a physician? (No) Does either of them examine patients or provide medical treatment to people? (No)
- Did ACOEM hold an open process to solicit potential contributors to the preparation of this paper? (No)
- Did ACOEM send a request to ACOEM members with expertise in this area to contribute or provide peer review?
- Are you aware of whether any other ACOEM Evidence-Based Statements or Position Papers have been authored by non-ACOEM members? By physicians whose principal specialty is something other than occupational medicine? (State: In fact, there are none that I'm aware of, other than yours)
- Do you find it unusual that ACOEM would change its policy on authoring of organizational statements for this one particular issue? Do you know how or why this came about?
- Which people specifically peer-reviewed your document? Are you aware of whether any of them have ever published any peer-reviewed articles concerning indoor air quality, mold, or related issues? (None have)
- What are the affiliations of Dr. Kelman? Dr. Hardin? (Global Tox)
- What is Global Tox?
    - What percentage of Global Tox work is involved in performing services on behalf of insurance companies, law firms, or companies involved in litigation? (100%)
    - Who is Corrine Robins (CIH—formerly worked for Gots)
    - What percentage of Dr. Kelman's work is devoted to insurance defense litigation consulting, including expert work? Dr. Hardin? How about plaintiff work—are you aware of whether either of these "toxicologists" ever assists plaintiffs? How about individuals

who have health problems or concerns related to mold exposure who are not in litigation?

- Who is Ronald Gots?
    - What percentage of Dr. Gots' work is devoted to work on behalf of law firms, insurance companies, and companies that are defendants (as opposed to plaintiffs) in any litigation? Same question for mold litigation?
    - Does Dr. Gots see patients or provide patient care? (No)
    - Does Dr. Gots have training or certification in Occupational Medicine? (No)
    - What is your relationship with Dr. Gots? How often and in what cases have you worked with him? (If he says none, he's lying.)
    - When was the last time you spoke or corresponded with Dr. Gots?
    - What is the relationship between Gots and Global Tox? (All Global Tox people came from Gots' firm in DC!)
    - Is it not true that Dr. Gots published an article urging occupational medicine physicians to "take a position" that mold is not a valid health hazard?
    - Is it not true that around the time that article was prepared, your group began working on the ACOEM document?
    - Were you approached personally by Dr. Gots or a member of one of his consulting firms to participate in the ACOEM paper?
    - Do you know if ACOEM aware that Global Tox is largely an insurance defense litigation firm?
- Do you consider it unusual or questionable that an organization such as ACOEM would develop such a paper without any direct contribution from, or solicitation of its own members or profession?
- Do you feel that your position paper comprehensively addresses the issue of adverse health effects associated with molds?

Knowledge of IAQ/Mold

- I understand that you have some 180 or so peer-reviewed publications in your career. Of those, how many other than the ACOEM paper concern the issue of indoor mold or indoor air quality? (None)
- In fact, I conducted a literature search of your work since 1993 and found NO papers having anything to do with mold, other than the ACOEM paper. Is it a fair conclusion that you have not published any original work in this area in the past 10 years?
- What is sick building syndrome? Are you familiar with the scientific literature on this topic? (Get him to list all articles and work)

- Are you familiar with the term "building-related illness?" Are you familiar with the scientific literature on this topic? (Get him to list all articles and work)
- What is the AIHA?
    - What is a certified industrial hygienist?
    - Are CIHs involved in mold exposure issues?
    - Are you familiar with the 1996 AIHA document, Field Guide for the Determination of Biological Contaminants? Have you read it?
    - Was this document cited in your ACOEM paper?
- What is ACGIH?
    - Are you familiar with the 1999 ACGIH document, Bioaerosols: Assessment and Control? Have you read it?
    - Was this document cited in your ACOEM paper?
- What is IICRC?
    - Are you familiar with the 2003 IICRC document, S520 Standard and Reference Guide for Professional Mold Remediation? Have you read it?
- What is ISIAQ?
- Are you familiar with the journal, Indoor Air? Do you subscribe to it? Do you read it regularly?
- Would you consider yourself knowledgeable in the medical literature concerning the topic of indoor mold?
- Are you familiar with an article by Kolstad et al in Epidemiologic Reviews, 2002 ("Do Indoor Molds in Nonindustrial Environments Threaten Workers' Health: A Review of the Epidemiological Evidence")?
- Are you familiar with an article by Hussman in Scandinavian Journal of Work, Health and Environment 1996 ("Health Effects of Indoor Air Micro-organisms")?
- Do you know if any of these documents were cited or reviewed in the preparation of the ACOEM paper?
- What is the California Department of Health Services? Are you familiar with their publications on indoor air quality?
- Do you have any training in the area of industrial hygiene?
    - What is an Anderson sampler?
    - What is a Burkard sampler?
    - What is MEA?
    - What is DG18?
    - What is a "positive hole correction?"

<u>Mold-Allergic Effects</u>

- What types (genus, species) of molds tend to grow on water-damaged building materials such as wet gypsum board?
- What types (genus, species) of molds are most commonly found outdoors in North America?
- Are the molds that grow on wet building materials the same as those found outdoors?
- Are allergen skin tests for mold specific for the types of mold that grow on wet building materials the same as those found outdoors?
- What is the false positive rate of allergen skin tests in people who don't have clinical allergies (allergic rhinitis)?
- Have any studies ever been published, to your knowledge, that have proved that people who have positive skin tests to molds actually have clinical allergic responses to that particular allergen(s)?

☐ ACOEM
☐ Sarcoma
☐ Gots article

Saxon

Cases
- Gary Small
- PTC
- Spectrum
- Lorber Perjvilly

Prof. activities
- D. Litigation - list
  % D., P.
  Is Tim Dunn? Agents?
- does UCLA know what his activities are?
- LIST all cases done in past 12 months — LIST ALL P. Cases!! (easy)
  $/fees; atty's

ACOEM
- How contacted? By whom
- Was Tim an open process?
- Is he an ACOEM member?
- What is the affiliation of the other authors?
- What is "Global Tox"?
  Does GT do 100% D. work?
- Who is Ronald Gots? Have you worked w/ him? What does he do?

How many peer reviewed papers by non-members of ACOEM are attached

Consent of interest

☐ Gots involvement in ACOEM

Knowledge of IAQ
- Past experience in IAQ & ACOEM?
- AIHA Field Guide 96
- ACGIH Bioaerosols 99 — BAS
- articles - List!
-

Mold - allergic?

Normal outdoor mold

Is mold that grows on